Other drugs acting synergistically with dopa include anticholinergics such as trihexyphenidyl hydrochloride and benztropine mesylate. Amantadine hydrochloride may have both short- and long-term benefits, particularly for tremor.

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Electrodiagnosis of Neuromuscular Disorders

ELECTRODIAGNOSTIC TECHNIQUES can be of help in the diagnosis, management, and prognosis of neuromuscular syndromes. Motor nerve conduction is determined by recording the compound muscle action potential, usually from a small intrinsic hand or foot muscle, while the nerve is stimulated at two locations. The distal latency reading is subtracted from the proximal latency reading and the difference divided into the distance measurement. Abnormal latencies and waveforms may indicate axonal loss or the presence of nerve conduction block resulting from demyelination and assist in locating the site of such abnormalities.

The F wave, resulting from electrical stimulation of the motor nerve, is an example of a "late response." It occurs when an antidromically conducted action potential reaches the motoneuron and is fired back out to the periphery. Prolonged latencies of late responses when nerve conduction is normal indicate the presence of proximal neuropathy, usually demyelination (acute Guillain-Barré syndrome). The F wave is conducted over the ventral root and is not a reflex. In some muscles, such as the soleus or flexor carpi radialis, it is possible to record the H reflex, which results from the activation of muscle spindle afferents. The H reflex uses the spinal monosynaptic reflex requiring the dorsal root. Disorders selectively involving the dorsal root can be detected by comparing the F-wave and H-reflex responses.

Sensory nerve action potentials are recorded from the skin or by near-nerve needle recording. Cerebral and spinal evoked potentials can also be recorded after nerve stimulation. These potentials often require signal averaging to record. The technique permits an investigation of disorders of the sensory nerve affecting large myelinated fibers and connections to cerebral cortex.

A repetitive stimulation of motor nerves while recording the compound muscle action potential (CMAP) is used to evaluate neuromuscular transmission. A decline in the amplitude of the CMAP at slow rates of stimulation indicates a postsynaptic defect; the reduced amount of acetylcholine produced following each nerve impulse finally falls below the level sufficient to activate some muscle fibers. A presynaptic defect, as seen in botulism or the Lambert-Eaton syndrome, causes an increase in amplitude with high rates of stimulation (> 10 Hz) as the release of acetylcholine is facilitated by increased levels of extracellular calcium associated with repetitive stimulation. The amplitude of the CMAP from a condition of rest is below normal.

A quantitative analysis of the amplitude, duration, and waveform of the motor unit action potential recorded by a needle electrode can be used to detect signs of motor unit loss, denervation, reinnervation—early and late—and muscle fiber loss. This technique can characterize the extent and severity of neurogenic atrophy as well as of myopathy.

The single-fiber electrode records the potentials of single muscle fibers from within a small electrode recording area. Two or more muscle fiber potentials can be recorded from within the same motor unit. By triggering the sweep of an oscilloscope with the single-fiber potential and delaying the display of action potentials, the synchrony of the firing of fibers and their density within the motor unit can be determined. Such studies are helpful in diagnosing disorders of the neuromuscular junction when the results of other studies are normal.

The latency and amplitude of reflexes can be used to assess central connections important for the reflex and the integrity of each limb. For example, the blink reflex can be used to evaluate an oligosynaptic ipsilateral response of orbicularis oculi muscle in response to stimulation of the trigeminal nerve, as well as a polysynaptic, bilaterally represented later response with pathways in the pons and medulla. Combined with motor conduction studies of the seventh nerve, lesions within the pons can be differentiated from those affecting one limb of the reflex.

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Nervous System Effects of Toluene and Other Organic Solvents

ORGANIC SOLVENTS are industrial compounds that produce neurologic syndromes related to both acute and chronic intoxication. Because hundreds of new organic compounds are introduced each year, many previously unsuspected toxic effects have been recognized. Solvents are highly lipophilic, which explains their distribution to organs rich in lipids, such as the brain. Although extremely high concentrations of all volatile organic solvents produce nonspecific effects, such as encephalopathy, many of these solvents produce relatively specific neurologic syndromes with lower level, longer term exposure. Long-term or persistent specific effects on the nervous system vary with the duration of exposure and whether the central nervous system (CNS) or the peripheral nervous system is primarily affected.

Long-term exposure to the aliphatic hydrocarbons, such as *n*-hexane and methyl butyl ketone, is typically associated with the development of peripheral neuropathy. Animal models developed using these compounds are classic examples of target-organ toxicity affecting the peripheral nervous system. The aromatic hydrocarbons, such as toluene, have been known to cause both acute and chronic CNS toxicity in humans for more than two decades, but because of the lack of an animal model of neurotoxicity and the small number of human cases, understanding the pathogenesis of the CNS effects of toluene has only come recently.

Toluene is the most widely used of the organic solvents